

HEART STARVATION.

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“THE PRACTITIONER’S HANDBOOK OF TREATMENT; OR, THE
PRINCIPLES OF THERAPEUTICS,”


ETC., ETC., ETC.

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HEART STARVATION.

OUR knowledge of the valvular diseases of the heart is now very fairly good, but our acquaintance with the heart as "a muscle" has not kept pace therewith. In the diagnosis of valvular diseases we have a murmur to guide us, not infallibly true, as being pathognomonic of organic change, but terribly suggestive; and giving us the locality of the disease, the direction of the blood current, and with it the changes to be looked for as consequent thereupon; indeed, telling us the malady, the natural history and progress of which are now fairly known. But, in the absence of a murmur, the evidences of cardiac asthenia leave us on the edge of bog in gathering darkness: there may be solid ground somewhere, but above all there is the bog, giving a sense of impending danger. The weird figure of "fatty degeneration" looms up in the darkness; either a reality or conjured up by the imagination. It is the mystery surrounding "fatty degeneration," its occult beginnings, its insidious course, the absence of precise indications; all combine to create a terror of it both in the minds of medical men and of the laity. Few medical students, when reading up the diseases of the heart, have kept quite free from a haunting suspicion that there might be some fatty necrosis going on in the structure of their heart-walls. But further familiarity tells them, in articulate language, that fatty degeneration is a senile change essentially; that it is a disease of advanced life *par excellence*, though sometimes found in comparatively early years. There is premature old age in the heart-walls, as well as elsewhere. It must be admitted that pericarditis, glueing the pericardium down over the coronary vessels, may lead to early decay in

the heart-walls, or the pressure of a syphilitic gumma may produce the same result. An atheromatous tubercle, blocking one or other coronary artery, may also lead to insidious necrosis of the muscular fibrillæ. But, as ordinarily met with, this fatty degeneration is the concomitant of atheroma; where the lumen of the coronary vessels is diminished, where the arteries themselves are tortuous, and consequently the blood current in them impaired and impeded; and where the aortic coats are affected and have lost much of their elasticity; and consequently the aortic rebound, the propelling force of the blood current into the coronary arteries, is lessened. With these arterial changes we find "the gouty heart;" first hypertrophied, and then, as its nutrition is interfered with, structurally impaired. Such, then, is the "natural history of the fatty heart." Its aspect is that of "old age"—physiological, if not always chronological old age. Here there is the doubtful, hesitating, uncertain step; the general infirmity of age. This is closely simulated by the muscular asthenia, the general debility of severe illness with impaired nutrition, found in younger persons. The general outline is the same; but one is a young person much debilitated, the other is a person unmistakably senile. The resemblance is diminished by scrutiny; careful observation demonstrates that the resemblance is more apparent than real. So with "fatty degeneration of the heart" and its double, "heart starvation;" the resemblance is greatest at first sight, and disappears on attentive scrutiny. A tottering old man, the subject of extensive senile changes, and a young or middle-aged man brought down by dysentery, may hardly be distinguishable at some distance, or to a casual glance; but, when seen closer, the resemblance fades away as the points of difference become more distinct. So it is with "fatty degeneration" and its double, "heart starvation."

We see epilepsy associated with a certain instability in the cortical cells of the motor area; we find it the outcome of hopeless structural changes. Yet the features of the epilepsy are much the same. We see a man stagger from drink, or from actual disease in the pons, or cerebellum. The mere act of staggering tells little. So with the evidences of cardiac asthenia; they tell us the heart is weak, but they do not instruct us as to the why of the cardiac

feebleness. Our diagnosis, then, is not cleared up by mere physical examination, nor even by the subjective phenomena, since these are the same; but by the associations of the enfeebled heart. It must be decided by the state of the individual, not by that of the heart; since this last is identical—identical so far as any human means, in the present state of our knowledge, can discern. Ordinary physical examination is dumb as to the means of discrimination. Where there is asthenia of the heart in a person carrying the senile aspect of the “fatty heart,” it is impossible to be certain about the diagnosis; and it is better to err (if err we must) on the safe side, and not to underestimate the gravity of a case; albeit that in doing so we add to the sum total of avoidable human misery by our error in a certain percentage of cases. If some cases carry with them a better prognosis than the symptoms seem to warrant, let patient and practitioner be thankful. Possibly some cases of aortic dilatation and aneurysm carry with them a less grave prognosis than the one usually given, namely, a hopelessly downward course; and diminution of the blood-pressure upon the internal surface of the sac leads to much recovery in the elastic arterial wall; but because this occurs in some cases caught at an early period and subjected to appropriate treatment, we are not warranted in looking upon either condition as being anything but very grave and serious. When the fortunate case does occur, it is to be regarded as just cause for rejoicing.

So in cardiac asthenia; only here the error of regarding “heart starvation” as “fatty degeneration” is one more often made than is quite creditable to the profession; for the difficulties of differentiation are not insuperably great in the bulk of cases. George Balfour wrote in 1876:—“The time is rapidly approaching when the knowledge of medical physics shall be so widespread as effectually to eliminate mere opinion, at least, from the diagnosis of diseases of the heart, an organ the condition of which we can ascertain in so many different ways, that with sufficient care we can almost be as certain of the state of its orifices, at least, during life as if we had it on the dissecting-table.” Yes; certainly Dr Balfour is nearly, if not quite right about “the state of its orifices, at least;” but about the state of its muscular walls we, *i.e.*, the profession generally, are,

and rightly so, much less confident. It is a far more difficult matter to ascertain, requiring a wider range of information. Sometimes accurate acquaintance with physiological function will tell us more than mere anatomically-furnished data, will often, indeed, speak out articulately when anatomical data are dumb. Disturbances of function cannot be estimated by individuals who have not learned systematically to think for themselves; whilst a fourth year student will commonly make a very accurate diagnosis, when this depends upon mere acute observation of physical signs. A certain familiarity with a foreign language is admitted to be requisite in order to enable the individual to think in it; a certain intimacy with physiology is essential to accurate reasoning upon data founded upon such knowledge.

This is a somewhat lengthy preamble, but it throws a quantity of side-light, not without its value, upon the subject of "heart starvation."

There are many conditions where "interstitial digestion" or "tissue nutrition" is impaired. We see at times a person whose muscles are well fed, but who is lean. Such is the condition of a man trained for some contest involving endurance; there is a maximum of muscle with a minimum of fat. Then at other times we see a man whose muscles are flabby, but who is well covered with fat. Here there is an opposite condition of nutrition, viz., a want of assimilation of albumenoids. A muscle well exercised attracts to itself more pabulum, as in the arm of the blacksmith and the leg of the ballet-dancer. Yes; so long as the blood is well fed and contains a sufficiency of albumen upon which the muscles, in their turn, may feed. But where a muscle is used very freely when the nutrition is defective, it wastes, or undergoes atrophy, as T. King Chambers has pointed out (*Clinical Lectures*). So, when the digestion of albumenoids is impaired, the two great muscles, the heart and diaphragm, in constant action, become more or less starved. This is very apt to occur when the liver is disordered seriously, so that the peptonized albumenoids which have passed from the gastro-intestinal canal into the blood, do not properly undergo their further and final transformations. A number of cases have occurred to me lately which have shown this associa-

tion of liver disorder with enlargement and debility in the cardiac walls—a debility against which digitalis and iron are simply impotent. All know (well, or ought to know) that when the liver is disordered the administration of iron never succeeds—often, indeed, makes matters worse. When the liver is put in order, then the tissue nutrition is improved, and the heart-walls become once more of normal power, and the symptoms of cardiac asthenia are relieved. Of the accuracy of the clinical phenomena there can be no question; whether the explanation is equally well founded is a matter upon which opinions may differ somewhat.

No one who has made the subject of digestion a matter of careful study can dispute the fact that the digestion of starch, albumenoids, and fats, is not always in strict proportion to each other; invariable and unvarying. It is not at all uncommon to find an unfilled artery in a corpulent person, even more often in a person well nourished, and therewith evidences of an anæmic brain, and breathlessness on exertion; and, further, with a deposit in the urine, most marked after meals, pale stools, and a foul taste in the mornings—the evidences of hepatic disturbance. At least I frequently see such cases. There may be actual enlargement of the liver, but at other times the viscus is of normal size. We do not yet know much of the liver in its early derangements, for they are not lethal; perhaps the disturbance does not produce any morbid change that can be detected by the microscope, even. Anyhow, we can only recognise disturbance of function. This is confined to the metabolism of albumenoids. Beyond difficulty in their actual digestion, hydrocarbons—starch, sugar, and fat—give us little or no trouble; but it is far different with the albumenoid elements of our food. From the time, as soluble peptones, they pass from the digestive tract into the blood, until the time of their reappearance as bile acids, and urine solids, their history is that of an underground river—we know it is somewhere; but its whereabouts is veiled from us.

The albumenoids give positive evidence of disturbance produced in their metabolism within the system by their actual presence, either as morbid products or natural products in excess—that chain of symptoms which are classed under indigestion, biliousness, or gout, as the case may be. Often there are evidences of arterial anæmia, and, blended

therewith, actual toxæmia from the products of disordered digestion, or normal nitrogenized waste in excess. That such is the case is proved by the fact of the disappearance both of the evidences of anæmia and of toxæmia together, when the liver is attended to rationally. The rational treatment is (1) to reduce the work of the liver by a dietary containing albumenoids in sparing quantities, and in easily digestible form; and (2) to sweep away the nitrogenized waste, and so cleanse the blood. A little proteid food well and thoroughly digested, gives more tissue pabulum than a meal rich in albumenoids; none of which are thoroughly and completely digested. Consequently upon such a dietary the tissues are actually better fed, better nourished; while there is a minimum of nitrogenized waste to be got rid of. Upon a dietary rich in albumenoids there is a maximum of waste, deleterious as well as useless, without tissue nutrition. This waste in the blood often raises the blood-pressure in the arteries, and so offers an abnormal resistance to the ventricular systole: sweep away the waste, prevent its further accumulation, and an asthenic ventricle recovers itself. Such is the rightful treatment of many a feeble heart-wall which remains unaffected by digitalis and iron, with a liberal dietary rich in albumenoids if, indeed, it does not grow actually worse under such management.

The following case is to the point:—

The Rev. Mr S. consulted me with his medical attendant, Mr Webster of Bristol, on May 22d, 1880. He had for seven preceding months been under the care of one of the most eminent members of the profession in town, without avail. He had a mitral regurgitant murmur, a dilated left ventricle, an enlarged liver, pale stools, and a deposit in his urine. He was debarred from work by asthenia and inability to exert himself. He had been well fed, and taken digitalis, strychnia, and iron, without any improvement. I looked upon it as a case where the assimilation of albumenoids was impaired, so he was put upon a diet containing little albumenoid material; while twice a week he was purged with a mercurial pill at night and a saline purgative next morning, to cleanse the blood of its nitrogenized waste. He was enjoined perfect rest, in order to ease the work of the heart. He had a little pot. brom. and digitalis to relieve the palpitation. On June the 16th the report is,

"The liver is decidedly improved, and more bile is secreted, and less lithates in the water; his pulse feels more vigorous, and there is a fair approach to something like rhythm in it." On July 16th he was "very much improved indeed. Heart steadier; murmur scarcely audible; liver much less."

Since then I have only had reports of him. On December 20th "he was looking remarkably well and happy. So far he has made a most marvellous recovery since he has been under your treatment. He has carried out your dietetic instructions almost to the letter, and the result has been most satisfactory. The condition of the heart is most decidedly improved; it flutters less, and gives him very little trouble at night-time."

The latest account from his medical attendant, April 6th, says,—"Our patient is now progressing most favourably; you have done wonders for him. The heart-sounds are decidedly clearer, and the rhythm more natural—more power altogether, and he looks better and is in good spirits."

If this case stood alone it would carry comparatively little weight with it. Another runs as follows:—

E. W., æt. 52, had also been under medical care some time without result, except getting worse. His pulse was 120 and irregular, and there was cardiac dilatation. His respirations were 24 when quiet. His liver was decidedly enlarged. His appetite was bad, but there were lithates in his water. There was distinct dropsy in both legs up to the knees. He was put upon an identical plan of treatment, except am. carb. for pot. brom., with a little digitalis. By December 31st he ate well, slept well, had no nocturnal dyspnoea; heart's action nearly regular; dropsy quite gone. His recovery has gone on unimpaired, and he presents the appearance of a perfectly healthy man. His complaint is this; "I cannot take a four mile walk, which I ought to be able to do," he writes.

In both these cases the improvement in the liver led to better assimilation of albumenoids, and consequent better tissue nutrition; and the heart and diaphragm both improved thereby, and were functionally stronger. With this improved nutrition the evidences of asthenia, the dyspnoea and palpitation on effort, both disappeared.

On March 23d this year Dr Macintosh of the Brompton Road asked me to see with him a stout man, weighing over 18 stone, who had had a very severe attack of pleurisy. When lying quiet in bed he was well and quite easy. As soon as he got up he suffered acute pain, especially at the insertion of the diaphragm. He had a foul tongue and an enlarged liver. He was dieted and purged at intervals, and had some am. carb., digitalis, and nux vomica. When seated in a chair, and leaning his arms upon the back of another chair—a favourite attitude with persons with confirmed heart disease, so as to take off the weight of the head and shoulders—he was fairly comfortable. Here, again, there was muscular asthenia, the result of impaired assimilation of albumenoids. The keen east wind, which produced so sadly potent an effect on the Earl of Beaconsfield, has affected this patient's liver and retarded his progress.

This recognition of cardiac asthenia as the result of malnutrition is no new matter. Thirteen years ago I attended an old gentleman for a series of symptoms pointing to fatty degeneration of the heart. The first sound was very feeble, and he had all the subjective phenomena of a fatty heart. With rest, proper food, and some digitalis, he improved. Instead of the feeble heart, he developed a fairly hypertrophied heart-wall, with a distinct obstructive aortic murmur. This heart was “starved” rather than “fatty.” He enjoyed several years of very fair health after this. It is only recently, however, that the subject has forced itself upon my attention in its full panoply of symptoms and associations; and still more as to the most rapid and effective means of treating it successfully.

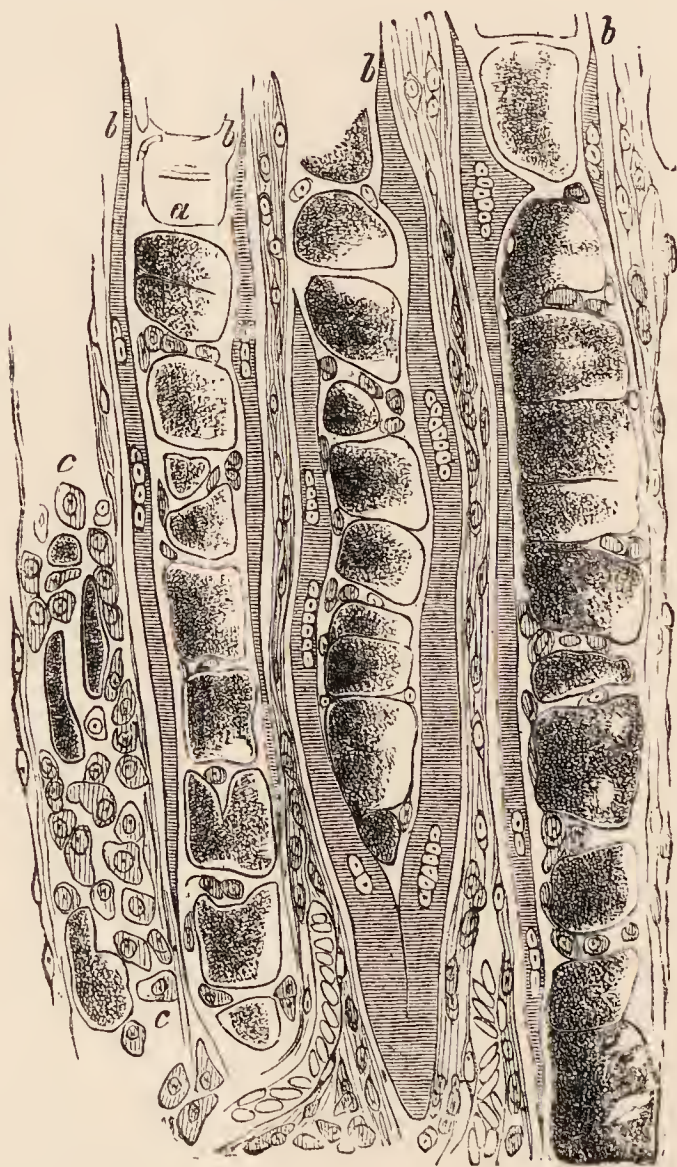
It is now time to review the symptoms and signs of “heart starvation,” contrasting them with those of “fatty degeneration.”

In his recent edition of his excellent work on *Medical Diagnosis*, my friend Dr Da Costa of Philadelphia writes of “fatty degeneration of the heart” so:—“Our power to recognise the change during life has not kept pace with our power to recognise it after death. There is as yet no sign discovered by which we can positively say that the dangerous disorganization of the muscular fibres of the heart is in progress. We may, however, suspect it if the signs of

weak action of the heart—feeble impulse and ill-defined sounds—coexist with oppression, with a tendency to coldness of the extremities, with a pulse permanently slow or permanently frequent and irregular, and be met with in a person who is the subject of gout, or of a wasting disease, or is very intemperate, or has arrived at a time of life at which all the organs are prone to decay.” So far the description will meet “heart starvation” from a long debauch without eating, or a wasting disease; just as well as “fatty degeneration.” He proceeds—“Something more than a suspicion is warranted if, in addition, there be proof of atheromatous change in the vessels, or of fatty degeneration elsewhere, such as an arcus senilis, or if it be ascertained that the patient suffers from pain across the upper part of the sternum, and from paroxysms of severe pain at the heart; that he sighs frequently; that he is put easily out of breath; that his skin has a yellow, greasy look; that he is subject to syncope or to seizures, during which his respirations seem to come to a standstill; and that he is liable to vertigo, or to be stricken down with repeated attacks having the character of apoplexy, save that they are not followed by paralysis.” Here we see that the aspect of “fatty degeneration” is distinctly “senile.” There are evidences of senile degeneration elsewhere; especially the atheromatous arteries—the natural concomitants of the fatty heart.

What there is in common with “heart starvation” is the feeble circulation, the cold extremities, the tendency to vertigo, the “attacks having the character of apoplexy, save that they are not followed by paralysis.” On March 17th last year I saw a lady lying on her bed unconscious and motionless, as she had lain for hours, and who lay there several more hours without alteration. Her ordinary medical attendant had kindly left a wooden boxful of leeches for my use. Knowing that she was worn out by long waiting upon a sister very ill, I distrusted the apoplexy view thoroughly, and shouted loudly at her. She opened her eyes, mentioned my name quite clearly, and then relapsed into her apoplectic state. Next day she got all right, except some cerebral confusion, and is now perfectly well. It was a case of “acute cerebral anæmia.” Such attacks are not, then, confined to fatty degeneration of the heart. The other signs of degeneration, as an arcus senilis

(Dr Da Costa pays myself the compliment of first describing the “true” as compared to the “false arcus senilis”) and a degenerate skin, are most important as factors in the diagnosis. But it must not be forgotten that “heart starvation” may show itself in a case where senile changes are undoubtedly present; where there may be possibly some degeneration of a proportion of the fibrillæ of the heart-walls, as in a case about to be related presently. There is no inherent impossibility of such coincidence. Beyond the matter of mere “heart starvation” lies that of the repair of degenerate heart-fibres, as given in a plate in my work on *The Heart and its Diseases, with their Treatment: including the Gouty Heart*, taken from Rindfleisch’s well-known work on *Pathology*, translated by the New Sydenham Society.



Acute Degeneration of the Heart (from Rindfleisch).

Here we see heart-fibres actually fattily degenerate after acute fever. We also see developing worm-like fusiform, or spindle-shaped fibres within the sarcolemma, growing up from embryonic

nuclei; and taking the place of the worn-out effete old fibres. This is a most instructive woodcut. It tells us that repair does go on in a decayed heart; when not due to such occlusion of the vessels as cuts down the blood-supply of a heart below the point of sufficient nutrition of the whole of the fibrillæ. It is this change in the coronary arteries found usually with the fatty heart which gives it its grave prognosis; because it forbids the possibility of repair. When the bloodvessels are not occluded, or not too far occluded, there exists no *a priori* reason against repair of the heart-walls by development of the embryonic muscular fibres already existing within the sarcolemma. It makes, indeed, the condition of the arteries the test for the gravity of the prognosis; as it rightfully is, and should be made. It is the extent of arterial change, rather than the information furnished by the stethoscope, which must tell us whether we have "fatty degeneration" or "heart starvation" to deal with; or, in some instances, probably both. Certain it is that we are not justified in regarding all cases of fatty degeneration—or, at least, what may fairly be termed "fatty degeneration"—as involving the hopeless prognosis which is correct and justifiable enough when there is advanced atheromatous disease of the arteries. To do so is to fall into the too common error of attributing to all affections of the heart, indiscriminately, the grave prognosis which properly attaches only to the cases where molecular decay is extensive and advancing. In an article in this Journal, February 1878, entitled "Some Conditions which simulate Organic Disease of the Heart," I pointed out several conditions which closely resemble organic changes. It is unnecessary to refer to them here. In one case the absence of impulse and the indistinctness of the heart-sounds had led the medical men into an error, which they readily enough acknowledged when Clifford Allbutt, F.R.S., pointed it out to them. Here too exclusive reliance upon the testimony furnished by the stethoscope, alone, led them astray. Very often careful examination of the radial pulse will correct an erroneous estimate formed from the character of the heart-sounds.

Now, as to the symptoms of dyspnœa, or breathlessness, often found in actual fatty degeneration of the heart. In the consideration of this symptom it seems surprising that more attention has

not been paid to the diaphragm. The most important muscle in the body, next to the heart, it seems to have escaped attention alike in the hospital ward and in the dead-house. Yet it must be subject to the conditions which control the nutrition of muscles. The same arterial degeneration which involves the heart-fibres must implicate the nutrition of the diaphragm in the tissue-wreck it works. So also in "heart starvation" the mal-assimilation of albumenoids must impair the functional activity of the diaphragm, and render it unequal to a sudden demand. So we get breathlessness upon effort, with frequent sighing. There is, too, a tendency to faintness, and attacks where the respiration seems to come to a standstill. Quite commonly there are attacks of nocturnal dyspnœa coming on in deep sleep. We know that in sleep the respiration becomes slow and deep. If the lowering of the activity of the nervous centres of the respiration, in the medulla, passes beyond a certain point, the blood becomes surcharged with carbonic acid, the natural stimulus to these said centres; and then these centres are roused to powerful and rapid discharges, which throw the respiratory muscles generally, the ordinary and also the extraordinary, into violent action. These vigorous respiratory efforts soon restore the blood to its normal state, by getting rid of the surplus carbonic acid; and then the usual quiet breathing is regained. Such is the history of an ordinary attack of nocturnal dyspnœa. When the circulatory organs are impaired and lacking in power, such attacks are rendered more likely to happen. When both heart and diaphragm are "starved," such attacks are found; just as in fatty necrosis of the fibrillæ of these two muscles. These attacks are of comparatively short duration contrasted with the true cardiac asthenia of over-distention of the right ventricle. The first cease naturally when the blood becomes purified and freed from the excess of carbonic acid, which excites the violent respiratory efforts; the latter, depending upon over-distention (the secondary elongation of Carpenter) of muscular fibre, require a longer period before they pass away. It is desirable to differentiate these two forms of nocturnal dyspnœa; as they belong to different conditions; and require each their appropriate treatment. The dyspnœa of failure of the respiratory centres is common in chronic Bright's disease. Such, then, are the linea-

ments of "heart starvation" which bear a strong resemblance to those of "fatty degeneration;" just as the features in serious syncope resemble those of a corpse in the first hours of death.

But "heart starvation" is not always associated causally with impaired tissue-nutrition, due to imperfect assimilation of albumenoids, with liver disturbance. It is found where there has been a great deficiency of sleep for some time. In some instances the cardiac asthenia takes the direction of dilatation. For instance, rather more than a year ago I was called in to see a woman of 40, of magnificent physique and unexceptionable family history; her father was alive and out, her mother an active old body of 80, who was bustling about, waiting upon her sick daughter. Here there was acute dilatation from over-exertion, want of sleep, and want of proper food. The patient was a nurse in a family where there were a number of children, to whom she was much attached. One after another they had scarlatina, and, almost single-handed, she nursed them day and night. When the last one was convalescent she broke down, exhausted with her efforts; prostration in bed, with a dilated heart, was the outcome of her devotion. Such acute dilatation as the result of over-exertion, especially when combined with insufficiency of sleep and comparatively little or no food, has long been familiar to me. It is one form of "heart starvation;" that form in which I have chiefly seen it in women. I have an impression, but will not put it more strongly, that exertion, over-effort, is an essential factor in the dilatation. Without such effort, in form and size the heart remains unaltered.

In 1874 I saw a gentleman, over 50 years of age, who was scant of breath on exertion, and who had a feeble radial pulse; and where the heart's sounds were very indistinct, while the impulse was lost altogether, partly from the dense chest-wall. He was a man of massive physique, but very gray for his years. He had been told that he was the subject of fatty degeneration of the heart by one of the most eminent of our authorities on the subject. He was taking much too little sleep; and longer hours of rest soon restored him to perfect health. Some years afterwards I saw him, and he was well and vigorous. Such cases are not very rare, and crop up from time to time.

Diagnosis.—The diagnosis of “heart starvation” is made by its concomitants, differentiating it from “fatty degeneration.” It is made by the grouping of the symptoms. Hayden justly says of the fatty heart, “The symptoms indicative of a fatty condition of the heart are individually of little value; but, combined in groups, they assume an affirmative significance.” So it is with “heart starvation;” always bearing in mind that “fatty degeneration” is a senile change, while “heart starvation” is a malady of earlier life: admitting that fatty degeneration may occur in rare instances in early life, and heart starvation be found in advanced life with the associations of the fatty heart—indeed, may occur in the sound fibrillæ of a heart already commencing to decay by fatty necrosis. The condition of the arteries is the most trustworthy guide in making the diagnosis, of any one individual factor.

There is, however, one condition of the heart which may easily be confounded with “heart starvation,” and that is “the irritable heart.” Indeed, I am myself in error in grouping these cases together in the second edition of my work on the *Diseases of the Heart*; being carried away from the correcter subdivisions of the first edition, by a series of cases of the irritable heart where muscular asthenia was a prominent symptom. Certainly they have many features in common, and in some cases it is impossible to say which is the greater half—the nervous irritability or the muscular adynamy. Some cases may be classed under either heading, and it is quite optional which shall be chosen. But they are obviously cases of heart disorder rather than heart disease—*i.e.*, there is no organic structural change, irreparable and irremediable. In the irritable heart there is a history of over-exertion with mental tension, leading to a neurosal condition. The action is notably increased beyond the normal on rising from the recumbent posture; it is quickened by emotion, and found mostly along with other evidences of the neurosal temperament. The distinction of the two has not quite escaped me, for I see it is written, “The effects of treatment will often help to clear up the case when the nature of it is obscure. Mere muscular failure in comparatively young and fairly well nourished persons, will be found usually to yield readily to rest, digitalis, and hæmatics; while the irritable heart improves

but slowly." But further experience tells me that the class of cases which lie outside the "usually" are those where there is mal-assimilation of albumenoids, and disordered function of the liver. Thus the special appropriate treatment must precede the iron and hæmatics (p. 399, 2d edition, 1879).

Prognosis.—This is a very grave matter, and needs much pains in its adjustment. On it hangs the future of the patient as a working member of society—perhaps the prospects of his wife and children. In the unmistakably fatty heart the outlook is dark, as dark as the oncome of night in a freshening south-western rain-storm. In heart starvation it is bright both as to result and as to time. It is in this last matter that it becomes so important to discriminate "heart starvation" from "the irritable heart," or to appraise correctly the proportions of the different factors when blended. Where the neurosal element is distinctly present, then the prognosis as to time is darkened. All neuroses possess an element of intractability. When there is the neurosal element distinct, the prognosis as to time is worse than when there is merely muscular asthenia from defective tissue nutrition. I may again quote in illustration thereof what is written elsewhere: "As to the prognosis, where the case was due to exhaustion of the sympathetic by hard marching and prolonged excitement, it was generally fairly amenable to treatment, especially when rest was attainable. But in other cases, where the disease assumed the character of a neurosis, the prognosis approached rather that of Graves's disease, and the case was intractable" (p. 390). Such was Da Costa's experience; such has been my own.

In both "heart starvation" and in the truly "fatty heart" there is a liability to attacks of angina pectoris vaso-motoria, *i.e.*, arterial spasm, when the blood is surcharged with nitrogenized waste. The prognosis of such attacks is in direct proportion to the extent of the fatty necrosis present.

Treatment.—This is a very important matter indeed, as correct treatment can alone enable the case to take the direction of restoration of functional power, by repair of the starved muscular fibrillæ. Digitalis and iron given indiscriminately will before long bring the treatment of heart-disease into disrepute; yet how the bulk of

medical practitioners are to be induced to study the features of each individual case, and to lay down a plan of treatment adapted to the requirements of each case? I confess it is not easy to see. The pressure on their time in large practice militates against such patient thought; and these cases are not common enough to make them quite familiar with the subject. It is necessary to remember first, that when the tongue is not clean, and the function of the liver is disordered, it is worse than useless to give iron (see "When not to give Iron," *Practitioner's Handbook of Treatment*, 2d edition). The liver and the assimilative processes must first be put to right, and then chalybeates may be given—but not before. As to medicines, it may be well to give a pill, the old-fashioned dinner pill, containing ipecacuan with a vegetable laxative. Ipecacuan is an hepatic stimulant of no mean potency; or iridin, or euonymin may be substituted. Then a little strychnia or digitalis may be added, according to the indications of the case. Twice a week, first thing in the morning, it may be well to give a dose of sulphate of soda with Rochelle salts, to sweep away a quantity of waste by the bowels, especially bile surplusage. This is the medicinal part of the subject. Now comes the dietary. A small quantity of albumenoids thoroughly digested furnishes more pabulum to the tissues, more material from which the tissues can be fed, than will a large meal, of which none or very little is completely digested. The tissues may be starved when the stomach is filled to repletion with food rich in albumenoid material. The food should consist of fish, fruits, fat, and farinaceous material — such dishes, indeed, as are given in *Food for the Invalid*; where a variety of suitable culinary receipts will be found. On such a dietary it will be seen that the patient gains weight as the nutrition of the tissues is improved. It is well, too, to aid the natural digestive efforts by giving pepsin, or the liquor pancreaticus advocated by Wm. Roberts in his recent *Lumleian Lectures*. After a certain improvement has actually taken place it may be well to proceed to administer chalybeates. The following case illustrates what may be attained by appropriate well-selected treatment even in a very unpromising condition.

Mr A., only 48, but looking much older, and presenting a decided

arcus senilis, consulted the late Dr Murchison some three years ago, who then said there was nothing organically wrong with the heart. When seen by me last autumn he complained of dyspnœa on effort, and at times without it; including attacks which came on at night in his sleep. He could not walk upstairs even slowly without breathlessness, extending to a paroxysm of dyspnœa if the greatest circumspection was not practised. There was decided cardiac dilatation, with irregularity, increased by effort. There were crops of petechiæ, indicating vaso-motor enfeeblement. There was much restlessness from the embarrassment of the respiration. The case seemed one of failure from fatty degeneration of the heart-walls. He was placed on the line of treatment given above. In a month he was calmer and quieter. The night attacks of dyspnœa had disappeared; but the breathlessness was easily induced by exertion, or mental annoyance. Then he returned to work, but grew worse; so he reduced the work, and again improved. A few weeks later the pulse was regular, the dyspnœa gone, and he could walk upstairs without inducing it. The appearance generally was much improved. He now had a little iron, with strychnia and digitalis in pil. al. et myrrh., with a little sulphate of soda and Rochelle salts in the morning once a week. The improvement went on steadily. When last seen he admitted having walked for considerable distances, and having "been upon his feet for two hours at a stretch without fatigue or discomfort;" while his wife said "he has been cutting up and down the steps of the Metropolitan Railway stations without complaint."

The progress of this case is most instructive. The evidences of cardiac asthenia are passing away; albeit that the general aspect of the case was that of the fatty heart. The heart-sounds are clearer and louder, the impulse better, along with the general improvement. Indeed, it looks as if there were new fibrillæ developing within the sarcolemma, taking the place of old effete fibres, and bringing with them an increment of power. The improved tissue nutrition is such that the diagnosis was doubtful, though it was also held by the eminent medical men under whom he had been before placing himself under my care: the reason assigned for coming to me being that they "lacked decision" in

their measures. Probably the case is one where actual fatty degeneration of some of the fibrillæ of the heart was blended with mal-nutrition of the fibres remaining structurally intact. Certain it is, that where there are distinct evidences of mal-assimilation of albumenoids it is well to treat decisively a case which even carries the broad lineaments of fatty degeneration—not to give it up in hopeless despair of doing any good. To do this is certainly to confirm the dark prognosis. But in order to secure all the improvement potentially possible in such cases of tissue-deterioration we must be familiar with what physiology is teaching us about the feeding of the body, the digestion of the various elements of our body, the function of the liver in relation to albumenoids; as well as the means of acting upon the liver, so much elucidated by recent physiological research. Physiology must guide our practice in the future. Anatomical or mere histological research has apparently told us all it has got to say. When it can lead to such an opinion as this,—“The fatal forms of progressive anæmia, to which hitherto no satisfactory cause has been assigned, are due to atrophy of the gastric glands,”—it is high time to critically examine its utterances. This state of atrophy of the gastric tubules may be found after death. But is it not the result, rather than the cause, of the pernicious anæmia—a case of getting the cart before the horse? Such an hypothesis ignores the trypsin of the pancreas as a digester of albumenoids; and is monstrously improbable on the face of it. If the digestion of our food is still to be made to rest so exclusively upon the stomach, our progress is blocked, or at least gravely impeded. Sounder, truer, more exact impressions of the digestive act are essential to a better acquaintance with the subject of tissue-nutrition and its disturbances, of which “heart starvation” is a part.